In the Specification

Please delete the entire section of the specification entitled "Brief Description of the Figures", beginning on page 4 and ending on page 6.

Please replace the paragraph beginning on page 31, line 27, with the following paragraph:

> RAGE-dependent binding to brain microvessels ~~(Fig. 1a)~~ and transport across the BBB ~~(Fig. 1b)~~ of human and mouse $A\beta_{1-40}$, and somewhat slower, but significant RAGE-dependent BBB transport of $A\beta_{1-42}$ ~~(Fig. 1b)~~ and absence of its significant binding to microvessels ~~(Fig. 1a)~~ were found in mice ~~(shown in Fig. 1a)~~ and guinea pigs. Aβ transport into brain was significantly inhibited by 65% to 85% by circulating α-RAGE IgC (5-40μg/kg) and abolished sRAGE. Several other molecular reagents including fucoidan (a ligand for the scavenger receptor type A), anti-β1-intergrin antibodies, or RHDS peptide (5-9 sequence of Aβ) did not affect either BBB transport or binding of Aβ ~~(Figs. 1a and b)~~. Although Aβ peptides were partially metabolized during their transport across the BBB (i.e., ≤ 50% for 10 min), significant and rapid RAGE-dependent neuronal uptake of circulating Aβ was observed after the BBB transport ~~(Fig. 1c)~~.

Please replace the paragraph beginning on page 32, line 14, with the following paragraph:

Transport of $A\beta_{1-40}$ across the BBB was associated with an early cellular stress response that preceded changes in the CBF. The expression of TNF-α mRNA and protein on different cells in brain parenchyma, including neurons and brain endothelium was evident after 15 min of transport of circulating Aβ across the BBB ~~(Fig. 2a)~~. Treatment with circulating sRAGE ~~(Fig. 2a)~~ or α-RAGE IgC abolished Aβ-induced TNF-α expression. Aβ transport across the BBB resulted in rapid neuronal expression of 1L-6 ~~(Fig. 2b)~~ and HO-1 ~~(Fig. 3c)~~, and these effects were abolished by either α-RAGE IgC ~~(Fig. 2b and c)~~ or sRAGE, supporting the concept that RAGE-dependent Aβ BBB transport initiates cellular stress in brain. RAGE-dependent Aβ-induced cellular stress was found either after cerebral arterial or systemic intravenous administration of Aβ, and persisted in brain for few hours. ~~Fig. 2d illustrates expression~~ Expression of TNF-α, IL-6 and HO-1 was observed in brain 2 hrs after i.v. administration of $A\beta_{1-40}$ at low nanomolar level.

Please replace the paragraph beginning on page 32, line 32, with the following paragraph:

Systemic administration of $A\beta_{1-40}$ (either human or murine) at low nanomolar concentrations resulted in time-dependent decrease in the CB, but did not affect systemic arterial blood pressure ~~(Fig. 3a)~~. Reductions in the CBF were detectable after 20-30 min of Aβ administration, and maximal decrease in the CBR was observed between 40-60 min. CBF changes were completely antagonized by circulating α-RAGE at 40 μg/ml ~~(Fig.~~

~~3b)~~. Aβ-induced cerebral vasospasm was antagonized by α-RAGE in a dose-dependent manner, was abolished by sRAGE, but was not affected by an irrelevant antibody ~~(Fig. 3c)~~.

Please replace the paragraph beginning on page 33, line 13, with the following paragraph:

~~Fig. 4a shows significant~~ A significant decrease in basal CBF values was observed in 9 months old Tg APPsw+/- mice compared to age-matched control mice as determined by laser Doppler flowmetry, and confirmed by quantitative autoradiographic analysis. There was no difference in the arterial blood pressure between wild type and TG APPsw+/- mice ~~(Fig. 4a)~~. Infusion of α-RAGE dramatically increased the CBF in Tg APPsw+/- mice ~~(Fig. 4b)~~, and the effect was maximal between 60-120 min after systemic administration of α-RAGE. An irrelevant IgC did not affect the CBF in Tg APPsw+/- mice, as indicated by moderate reduction is expression of TNF-a, IL-6 and HO-1 ~~(Fig. 4c)~~. Expression of RAGE on brain microvessels was enhanced in Tg APPsw+/- mice ~~(Fig. 4d left)~~, and increased vascular expression of RAGE was associated with accumulation of Aβ in AD brains ~~(Fig. 4d right)~~.